PEDIATRIC SURGERY

General Principles

Infants and children, in general, show major differences from adults. Their body systems have yet to develop and their full physiological functions and reserves are small. Thus it is easy for a small change to produce organ failure in one system; which may lead to failure of other systems.

CHAPTER CONTENTS

- General principles
- Congenital diaphragmatic hernia.
- Oesophageal *atresia*
- Congenital hypertrophic pyloric stenosis
- *Intestinal obstruction*
- *Childhood tumours*

Rate of change. Children are very fragile; a child can be moribund from meningitis within 24 hours or become dehydrated from gastroenteritis to the extent of peripheral circulatory failure in a day.

Temperature regulation. The small baby is easily affected by changes in environmental temperature. This is due to:

- Thinner layer of subcutaneous fat.
- Inadequate vasoconstriction of cutaneous vessels in response to cold.
- Absent or rudimentary shivering and sweating mechanisms
- Incomplete myelination of the CNS including the central heat regulating centre in the hypothalamus. As a result, all premature babies and most neonates will require incubators or at least wrapping of limbs and body with cotton wool to minimize heat loss.

Body fluids and electrolytes. 80% of the full-term baby's weight is water. This drops to 60% in adults. Maintenance of fluid and electrolyte balance is vital in the neonatal period. If oral intake is not feasible IV daily fluids are given.

- Daily fluid maintenance is given in the formula of 10% glucose in 0.18 percent saline. Potassium chloride (20 mmol/L) is added once urine flow is established.
- The fluid requirement of the neonate may be calculated according to the scheme in table 37.1.
- All fluid losses, for example, nasogastric aspirate and ileostomy losses are replaced with normal saline with potassium chloride (20 mmol/L).
- The above are merely guidelines and each case must be assessed individually with repeated electrolyte estimations.

Renal adjustment takes hours to days. As with adults and even more quickly, a baby may develop respiratory or metabolic acidosis or alkalosis. A simple formula that can be used to estimate the amount of bicarbonate needed to infuse in the first instance in the common condition of metabolic acidosis is: Number of mmol **HCO_3 = Base deficit X Wt in Kg/3**. However, proper correction of electrolyte and acid-base imbalance necessitates frequent monitoring of blood values several times daily.

Blood volume. In a neonate, the blood volume is about 80 ml/kg body weight (70 ml/Kg in adults). Thus a 3.5 kg neonate will have 280 ml total blood volume. Three blood stained swabs during surgical operation will contain 30 ml of blood which constitute 10% of the total blood volume. Such loss is significant in neonates but very negligible in adults. Blood transfusion may be resorted to in restricted indications, e.g. blood loss, haemolytic

anaemia or severe anaemia. A general formula of 10 ml/kg of blood or 5 ml/kg of packed RBCS is usually adequate.

Respiratory system. The lungs have sufficient capacity for postnatal survival at about 24 weeks gestation. After delivery, the lung matures rapidly but only about half of the alveoli are present at birth. It is worth remembering that the most energy consuming activity of the neonate is breathing. This explains the rapidity th which respiratory failure develops in babies with pneumonia.

	Maintenance IV fluids/day
Neonate	60 ml/kg to be raised by l0 ml/kg daily up to l00 ml
<10kg	100 ml/kg
> 10 kg	100 ml/kg/ for the first 10 kg + 50 ml for each additional kg of body weight

Hepatic function. The newborn liver is still immature, The most common example of this is the physiological jaundice that is brought about by the underdeveloped glucoronyl transferase system. Many other enzymes and functions are also involved, e.g. albumin, clotting factors and vitamin K. The liver acts also as a carbohydrate store in the form of glycogen. Even the full-term baby can not withstand starvation more than 6 hours without running the risk of hypoglycaemia.

Feeding. Feeding may be by the following methods, breast, bottle, nasogastric tube, gastrostomy, trans-gastric duodenal or jejunal tube or intravenous fluid therapy. If the baby cannot tolerate enteral feeding for more than a week, total parenteral nutrition (TPN) is mandatory.

Central nervous system. Myelination within the central nervous system is poorly developed at birth. The swings in heart and respiratory rate and temperature regulation are examples of the immature negative feedback loops at birth. The process of myelination is not completed until the late teens. The development milestones of childhood are related to the gradual maturation of the cortical connections within the brain.

Pain relief and analgesia. It was commonly stated that infants do not feel pain because of lack of central myelination. There is enough evidence to suggest that babies and small children produce physiological response to pain. It is also important to remember that the blood brain barrier is less efficient until 2 years of age due to incomplete myelination. As a result, fat-soluble drugs will permeate more freely into the cells in the brain in this age group. Thus opiates may produce respiratory depression at low doses. For example, morphine is given to adults in a dose of 0.2 mg/kg 6-8 hourly, but in the small infant 0.1 mg/kg 6-8 hourly is enough.

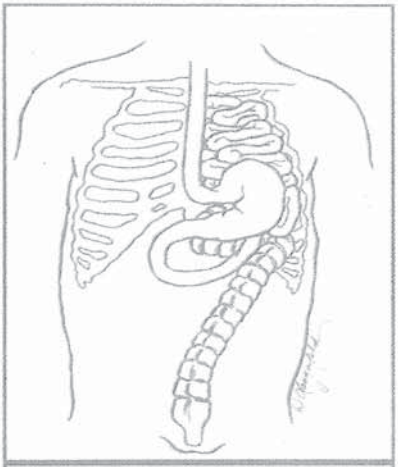

Fig. 37.1. Bochdalek hernia.

Congenital diaphragmatic hernia (CDH):

Any newborn presenting with respiratory distress, in the absence of heart disease, and a scaphoid abdomen should be considered as having congenital diaphragmatic hernia until proved otherwise. The incidence of CDH is 1:4000.

Embryology and aetiology:

Important developmental events:

1. Fusion of the septum transversum and the pleuroperitoneal folds occurs bilaterally during the 8th week of intrauterine life (right side before the left).

2. The intestine returns into the abdomen for rotation and fixation at the 10th week.

Consequences of diaphragm maldevelopment

- If diaphragmatic development is incomplete by the 10th week, the pleuroperitoneal hiatus (foramen of Bochdalek) persists and intestinal loops may herniate into the chest resulting in posterior diaphragmatic hernia. This will result in compression of the lung interfering with its development. As a result, there may be severe hypoplasia of the lung.
- Failure of fusion between the costal and sternal parts of the diaphragm will result in anterior (Morgagni) diaphragmatic hernia.
- Eventration of the diaphragm is a rare condition characterized by severe attenuation of its muscular and fibrous parts. Thus, the diaphragm is very flabby and protrudes into the pleural d'avity.

Types of diaphragmatic hernia

1. Posterior or Bochdalek hernia (Fig. 37.1) constitutes 90% of cases (left side more than right, Lt:Rt = 5:1).
2. Anterior or Morgagni hernia,
3. Hiatal hernia (through the oesophageal hiatus).
4. Eventration of diaphragm (not a true hernia).

Pathology:

The main problems in diaphragmatic hernia are:

1. Ipsilateral lung hypoplasia results in cyanosis and pulmonary hypertension, and leads to right to left shunt.
2. Contralateral lung compression by the abdominal contents.
3. Ischaemia of the herniated abdominal contents.

Clinical features:

Antenatal diagnosis:

- Maternal polyhydramnios raises suspicion.
- Antenatal ultrasound is diagnostic.

After birth:

In severe cases with marked pulmonary hypoplasia symptoms appear immediately after birth and include:

- Therei is respiratory distress, and gasping.
- General examination reeals cyanosis and tachypnea.
- Chest examination reveals that the heart sounds are better heard on the right side in case of the common left sided hernia. Intestinal sounds may be rarely heard on the chest.
- The abdomen is scaphoid.

Mild cases may present late by recurrent chest iinfections or by accidental auscultation of bowel sounds on the chest.

Investigations:

- Chest X-ray reveals gas shadow of the stomach or bowel in the thorax (Fig. 37.2).
- Blood gases to assess the degree of hypoxia.
- CT scan to the chest

Differential diagnosis:

Other causes of neonatal respiratory distress, e.g. congenital heart disease, oesophageal atresia, aspiration, and hyaline membrane disease

Fig. 37.2. Chest X-ray of a neonate with Bochdalek hernia. Intestinal loops that are filled with gas are seen inside the left side of the chest. The heart is pushed to the right.

Treatment:

Preoperative care (in a paediatric surgical center)

- Naso-gastric tube insertion to deflate the stomach.
- Endo-tracheal tube intubation and ventilatory support to correct the hypercapnea and acidosis.
- IV fluids
- Incubator to keep body temperature.

Operation:

Through an abdominal approach, the herniated abdominal contents are reduced and the defect is closed directly or a mesh is used if the defect is too big.

Post-operative care:

Continued ventilatory support is usually required. Honeymoon period lasts between 2-5 days postoperatively after which pulmonary hypertension develops and causes right to left shunts and eventually death may occur in about 50% of cases.

Modern trends in treatment:

- Intra-uterine surgical correction has limited success.
- ECMO (Extra Corporeal Membranous Oxygenation) is used in some centres to facilitate oxygenation until the lung matures.
- Nitric Oxide and toulazoline are used in some centres as they induce pulmonary vasodilatation.

Oesophageal atresia

A newborn presenting with repeated accumulation of frothy saliva should be considered to have oesophageal atresia until proved otherwise.

The Incidence is 1:4000. Males are affected more than females and the condition is more common in white than black races.

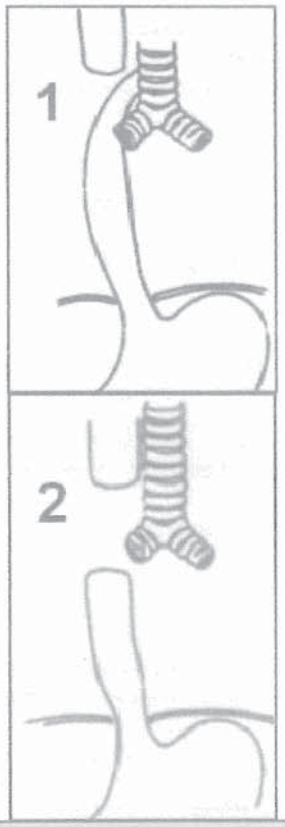

Fig. 37.3. Oesophageal atresia.
1. With tracheo-oesophageal fistula (common).
2. Without fistula (rare).

Aetiology

The condition is due to a defect in the division of the proximal fore-gut into ventral tracheobronchial tube and a dorsal oesophageal tube.

Pathology

Types: (Fig. 37.3)

1. The commonest type of oesophageal atresia is proximal blind atresia with distal tracheo-oesophageal fistula (87%).
2. Rare types include atresia without fistula (7%).

Complications:

1. As a result of proximal oesophageal atresia, the saliva and milk (if the baby was fed) will fill the upper pouch then trickle into the trachea and mouth causing bronchopneumonia and frothy saliva.
2. As a result of the distal tracheo-oesophageal fistula, the acidic gastric juice will regurgitate through the trachea into the lungs causing acid pneumonia which if persists is fatal.
3. Failure of feeding will lead to dehydration and hypoglycaemia.

Association anomalies:

The acronym "VACTERL" helps to remember a group of congenital anomalies that are commonly associated.

Vertebral (spina bifida with its various types, chapter 57).
Anorectal.
Cardiac (chapter 28).
Tracheal.
Esophageal.
Renal (30%, chapter 43).
Limb anomalies.

Clinical features:

Antenatal diagnosis:

- Maternal polyhydramnios may be present as in any case of antenatal intestinal obstruction.
- Antenatal ultrasound may show a dilated upper pouch of the oesophagus.

At birth:

1. Repeated accumulation of frothy saliva after proper resuscitation.
2. Attacks of chocking and cyanosis.
3. Pneumonia develops rapidly if the condition was not diagnosed before or immediately after birth.
4. Abdominal examination reveals abdominal distension with air if the fistula is large enough. If oesophageal atresia is suspected oral feeding is strictly prohibited until the condition is excluded.

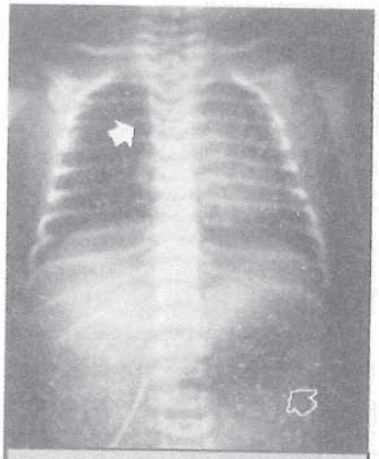

Fig. 37.4. Plain X-ray in a case of tracheo-oesophageal fistula. White arrow indicates the site of arrest of a nasogastric tube. Transparent arrow points at excess gastric air that indicates the presence of a fistula.

Investigations:

1. Catheter test. Insert a radioopaque catheter into the nose down to the oesophagus. If the catheter is arrested at 10cm, this denotes oesophageal atresia.
2. Plain x-ray of the chest and abdomen after inserting a nasogastric tube. This will confirm the presence of atresia of the oesophagus. The presence of a gastric air bubble will confirm the presence of a fistula (Fig. 37.4).
3. Other investigations to evaluate associated anomalies, e.g., renal ultrasound or echo-cardiography.
4. Blood gases to assess the need for assisted ventilation.

Treatment:

Early diagnosis before starting oral feeding is crucial to avoid pneumonia. The treatment is by urgent surgery in a paediatric surgery centre.

Preoperative care:

- Oral feeding is prohibited.
- Continuous low pressure suction of saliva.
- The infant is kept in an incubator.
- IV. fluids and antibiotics.
- Transfer to paediatric surgery centre.

Operation:

- Atresia with a fistula. Right thoracotomy, ligation of the fistula, and primary anastomosis of oesophageal ends should be tried.
- Atresia without fistula. Usually there is a long gap which makes immediate anastomosis difficult. A gastrostomy is, therefore, done at birth. Further surgery to restore oesophageal continuity is to be planned 6 months later.

Congenital hypertrophic pyloric stenosis (CHPS)

Any neonate presenting with projectile, non bile stained vomiting not responding to antiemetics and associated with hunger should be considered to have CRPS until proved otherwise.

Incidence:

- 1/400 neonates.
- M:F=4:1
- More in first born babies.
- Familial predisposition. The condition is more frequent in babies whose mothers had pyloric stenosis as neonates.

Aetiology:

No definite cause has been identified. The following factors, however, may be implicated.

- Improper innervation of the pyloric sphincter.
- Reduced nitric oxide synthesis.
- Increased growth hormone level.

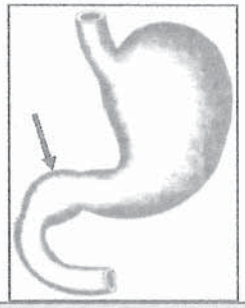

Fig. 37.5. Hypertrophied pyloric sphincter.

Pathology:

- At birth the pylorus is normal, but within 2-3 weeks, there is gradual progressive hypertrophy of the circular pyloric muscles (Fig. 37.5).
- This results in gradual occlusion of the gastric outlet. The baby continues to suckle but the milk cannot pass beyond the stomach leading to projectile vomiting.
- Recurrent, frequent vomiting results in loss of fluids, acid, chloride and salt. Thus blood chemistry often shows hypochloremia, hyponatremia, hypokalaemia and alkalosis.
- The stomach contracts trying to push its contents against the pylonc resistance, this explains the visible gastric penistalsis usually seen over the abdominal wall.

Clinical features:

Symptoms:

- Vomiting is the main feature. Vomiting is projectile, and non- bilious. It usually starts 2-3 weeks after birth. It does not respond to antiemetics. Sometimes the vomiting may be delayed. After vomiting the infant is very hungry and cries for its feeds.

- Firm stools.
- Loss of weight.
- In 10% of cases the infant has haematemesis due to gastritis or oesophagitis.

Signs:

- General examination, Dehydration is present in neglected cases.
- Abdominal examination:
    - The hypertrophied pyloric muscle is usually felt as an olive-like mass, Feeling this mass is diagnostic of the condition. The olive mass is felt in the transpylonic plane to the right of the right rectus muscle, better when the stomach is empty.
    - Test feed. The mother is asked to feed the baby, and then the examiner inspects the abdomen for visible peristalsis passing from left to right and palpates the abdomen for the olive like pyloric mass which is felt in the epigastric region on the undersurface of the right lobe of the liver.

Fig. 37.6. Barium meal showing elongated narrow pyloric canal

Differential diagnosis:

- Other causes of neonatal vomiting.
- Gastroenteritis.
- Gastro-oesophageal reflux.
- Feeding problems.
- Intestinal obstruction.

Investigations:

- **Imaging** is required only if the olive tumour is not palpable.
    - **Ultrasound**. This is the imaging modality of choice. CHPS is diagnosed if the thickness of the pyloric muscle is more than 4 mm.
    - Barium meal is performed if the facilities and experience in paediatric ultrasound are not available. The radiologtcal features are:
        - Dilated stomach.
        - Exaggerated peristalsis.
        - The pyloric canal is elongated and narrow (Fig. 37.6).
        - Delayed gastric emptying.
- **Laboratory investigations** (serum electrolytes): These are crucial to assess electrolyte deficits as these babies usually suffer from hypochloraemia, hyponatraemia, hypokalaemia and alkalosis.

Treatment:

Treatment is surgical. It requires proper preoperative preparation.

Preoperative care:

- Correction of dehydration and electrolyte deficits by IV fluids before surgery.
- Stop oral feeding.

It usually takes 24 hours to rehydrate the baby to be fit for surgery.

Operation. Ramstedt pyloromyotomy (Fig. 37.7):

- General anaesthesia.
- The abdomen is entered through a small transverse right upper abdominal incision.

- The hertrophied pylorus is grasped between the index and the thumb and a myotomy is performed until the mucosa of the pylorus bulges.
- If the mucous membrane is injured, the injury should be sutured and reinforced by an omental patch.

Fig. 37.7. Ramstedt's operation.

Postoperative care:

Oral feeding can be resumed after a few hours.

Prognosis. The results of this operation are excellent and the mortality should be around zero.

Intestinal obstruction in neonates, infants and children:

Main causes:

- **Duodenal obstruction:**
  1. Atresia.
  2. Malrotation with bands or volvulus (volvulus neonatorum).
  3. Annular pancreas.

1. **Jejunal and Heal obstruction:**
   1. Volvulus neonatorum.
   2. Atresia.
   3. Meconium ileus.
   4. Peritoneal bands.
   5. Strangulated inguinal hernia.
   6. Intussusception (chapter 36).
   7. Postoperative peritoneal adhesions, e.g., following appendicectomy.
2. **Large bowel obstruction:**
   1. Hirschsprung's disease.
   2. Anorectal anomalies.
   3. Meconium plug syndrome.
3. Necrotizing enterocolitis
4. Adynamic intestinal obstruction, e.g., due to hypokalemia or pneumonia.

Early detection:

1. Ante-natal. Polyhydramnios raises the suspicion of high gut obstruction.
2. During routine assessment of a neonate, that has just been delivered, a tube is passed down the oesophagus for aspiration of secretions and to exclude oesophageal atresia. The tube is further passed down to the stomach. A neonate with bilious gastric aspirate more than 25ml should be considered to have intestinal obstruction until proved otherwise,
3. Any child presenting with persistent, bile-stained vomiting should be considered to have intestinal obstruction until proved otherwise. Waiting for this stage, however, carries the risk of aspiration pneumonia.

Clinical features:

As for adults, the cardinal signs of intestinal obstruction are 2V, 2C, 2D, i.e.,
2 "V"s Vomiting and Visible peristalsis.
2 "C's Colics and Constipation (absolute).
2 "D's Distension and Dehydration.

1. Vomiting is the cardinal symptom of all types of intestinal obstruction. It may be early in cases of proximal intestinal obstruction, or late in distal intestinal obstruction. Greenish or brown faeculent vomiting is diagnostic of intestinal obstruction.
2. Visible peristalsis usually indicates mechanical obstruction; They are:
    a. In the epigastrium if obstruction is gastric or duodenal.
    b. Central abdominal in small intestinal obstruction.
    c. Peripheral abdominal in colonic obstruction.
3. Colics are manifested in babies by crying and withdrawing of legs.
4. Constipation. Failure to pass meconium or stools for more than 24 hours usually indicates intestinal obstruction in infants and neonates.
5. Distension follows the same pattern as visible peristalsis i.e. It is:
    a. Epigastric in gastric or duodenal obstruction.
    b. Central abdominal in small intestinal obstruction.
    c. Peripheral abdominal in colonic obstruction. However, if distal intestinal obstruction is presenting late, there may be diffuse severe abdominal distension.
6. Dehydration

Management

First aid

1. **Insertion of 3 tubes**
    a. Nasogastric tube.
    b. IV. access.
    c. Urinary catheter or urinary bag.
2. Fluids and drugs
    a. Ringer's lactate solution 10-20 ml/kg over the next hour according to the degree of dehydration.
    b. Antibiotics to cover both aerobic and anaerobic organisms, e.g. peflicillin, gentamycin and metronidazole.
3. Electrolytes and acid base fine adjustments according to blood chemistry.

Confirm diagnosis

1. Plain abdominal x-ray: This is the first and most important investigation. It may reveal the presence of distended bowel loops ad the presence of more than three fluid levels. The gas pattern usually helps to identify the cause of many problems.
    a. Double bubble in duodenal obstruction.
    b. Free air in peritoneum in intestinal perforation
    c. Calcification in intra-uterine perforation.
    d. Ground glass in meconium ileus.
    e. Intra-mural gas in necrotizing enterocolitis.
2. Barium meal may be needed to diagnose incomplete intestinal obstruction and mairotation.
3. Gastrografin enema is particularly helpful in Hirschsprung's disease and meconium ileus. In the latter it may also be therapeutic.
4. Rectal biopsy may be needed for suspected Hirschsprung's disease (colonic aganglionosis). Definitive treatment

Individual causes of paediatric intestinal obstruction

Volvulus neonatorum

- The condition is caused by incomplete rotation of the midgut that should occur during intra-uterine life (chapter 36).

- The baby is born with a free midgut having a narrow-based mesentery, and is, therefore, liable to twist around its mesenteric axis. The result is strangulation obstruction of the whole midgut.
- In addition, there is usually a fibrous band ([add's band) that extends from below the liver to the left to the caecum which lies in the epigastrium. This can obstruct the duodenum.
- Urgent surgery is needed to untwist the gut, to divide the band and to broaden the base of its mesentery by further displacement of the caecum to the left (Fig. 37.8).

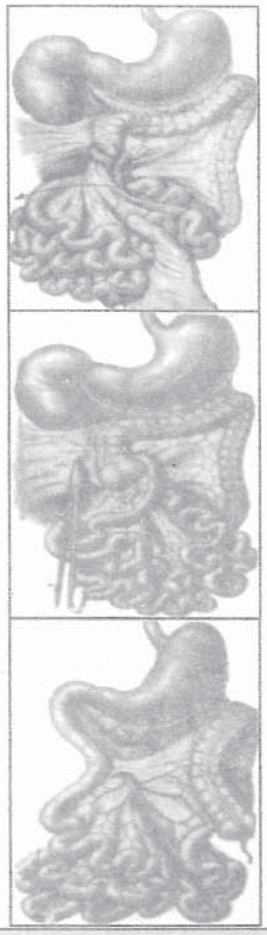

Fig. 37.8. Volvulus neonatorum. Untwisting, division of band of Ladd and displacement of the caecum to the left.

Jejunal and ileal atresia

- Jejunal and ileal atresia are due to obstruction of mesenteric arteries in utero.
- The atretic segment of bowel may be a cord like structure (Fig. 37.9) or may be completely absent.
- In jejunal atresia there is early bilious vomiting but distension is lacking.
- In ileal atresia abdominal distension is marked while vomiting may be delayed.
- In both conditions some meconium may be evacuated.
- Plain abdominal X-ray reveals distended bowel loops with fluid levels.
- Urgent surgery is required. Resection of the atretic segment is done with bowel anastomosis.

Meconium Ileus:

- There is obstruction of ileum in a neonate due to thick inspissated meconium.
- The condition occUrs in 10% of patients with cystic fibrosis (mucoviscidosis).
- Gastrografin enema may be diagnostic as well as therapeutic. Gastrografin absorbs water that may dissolve the thick meconium.
- Ileostomy may be needed and may be life saving.

Peritoneal bands:

Peritoneal bands, e.g., band of [add, or bands in relation to Meckel's diverticulum may compress the lumen or precipitate volvulus.

Fig. 37.9. Ileal atresia with proximal dilatation.

Strangulated inguinal hernia:

Strangulated inguinal hernia is a common cause of partial or complete intestinal obstruction during the first 6 months of life. Urgent surgery is required.

Hirschsprung's disease (aganglionic megacolon):

Any newborn presenting with delayed passage of meconium for more than 24 hours should be considered as having Hirsch- sprung's disease until proved otherwise.

Incidence is 1:5000
- M:F 4:1.
- There is familial incidence in 6% of cases.

Aetiology:
- The disease is due to absence of ganglia in the submucous and intermucular plexuses. The disease starts in the upper part of the anal canal and extends proximally to the recto sigmoid area in 80% of cases (typical case) or it may involve variable lengths of the colon or even the whole colon.
- The affected segment of bowel is spastic and narrow. The normally ganglionic colon proximal to it is markedly dilated (Fig. 37.10).

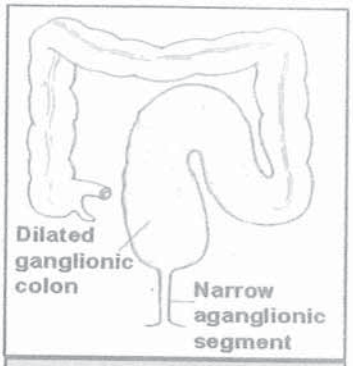

Fig. 37.10. Hirschsprung's disease.

Clinical features:
- The problem starts since birth. There is delayed passage of meconium. The mother notices that the infant is suffering from chronic constipation. Defecation occurs every few days and only after insertion of a suppository or doing an enema.
- Progressive abdominal distension develops.
- Sometimes, chronic intestinal obstruction culminates into acute obstruction with severe abdominal distension, absolute constipation and there may be vomiting.
- A severe complication called obstructive enterocolitis is liable to occur. During the affack the patient develops pyrexia, abdominal distension and diarrhoea.
- Delayed growth and development.
- Digital rectal examination reveals an empty rectum and when the finger is removed, it is followed by a sudden gush of gases or stools.

Fig. 37.11. Barium enema of congenital megacolon showing a narrow aganglionic distal segment (arrows) and marked proximal colon dilatation.

Investigations:
- Plain abdominal x-ray reveals large bowel obstruction.
- Barium enema needs to be done without preparation and a small amount of barium is administered. It usually shows the narrowed aganglionic segment, followed by a dilated normal ganglionic bowel (Fig. 37.11). Its diagnostic accuracy is 80%.
- Rectal biopsy. Previously full thickness rectal biopsy was recommended, but nowadays only a small segment of the mucosa and submucosa is taken by a special forceps. Ganglion cells in the submucous layer are absent.
- Anorectal manometry reveals failure of relaxation of the anal phincters in response to rectal distension.

Treatment:
- Patients presenting by intestinal obstruction
  - Initial conservative treatment by nasogastric tube suction, IV. fluids and repeated colonic washouts with saline.
  - If the obstructiion is relieved, prepare the patient for later elective surgery.
  - If the obstruction is not relieved perform urgent colostomy.
- Patients without obstruction require elective surgery.
  - Surgery is ideally performed at the age of 6-9 months.

- Definitive surgery for Hirschsprung's disease entails com plet excision of the aganglionic segment of the bowel up to 4 inches above the funnel and then anastomosis of the nor ma colon to the lower half of the anal canal.

Anorectal malformations:

Types:

- High anomalies where the rectum ends above the level of the pelvic floor and it is either a blind pounch or more com monl there is a fistulous connection to the bladder or pos terio urethra (Fig. 37.12) in males or to the vagina in fe males.
- Low anomalies where the rectum has passed through the pelvic floor and usually opens into an ectopic site anterior to the normal anus position. A fistulous connection may pass to the vestibule in females or to the perianal skin in males (Fig. 37.13).

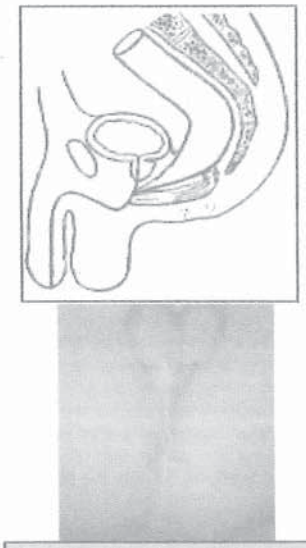

Fig. 37.12. High imperforate anus.

Incidence 1:5000.

- In high anomalies M:F2:1.
- In low anomalies M:F = 1:2.

Associated anomalies:

VACTERL. These associated defects are often serious and adversely alter the prognosis for the infant. They are more common in the high than in the low types.

Clinical features:

Examination of the perineum:

- Look for the presence of anus, its size and site.
- Look for the presence of an anal dimple.
- If there is an impulse on crying at the site of the anus, it denotes a low anomaly.
- In ectopic anus, there is a subcutaneous fistulous tract full of meconium.
- Meconium at the tip of the penis denotes a listula to the bladder or urethra
- If a thermometer can be introduced into the anus for more than one cm, this excludes rectal atresia.

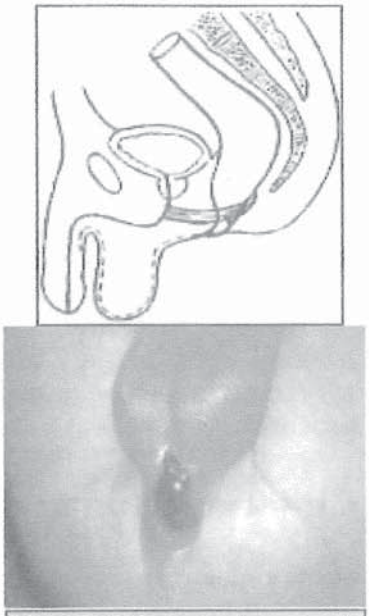

Fig. 37.13. Low imporforate anus. In this neonate a fistula opens by a tiny opening at the base of scrotum.

Abdominal examination:

There may be evidence of intestinal obstruction.

General examination:

To exclude associated congenital anomalies.

Investigations:

- Plain X-ray in the inverted position (Invertogram). The idea of this radiological test is that 24 hours after delivery, enough gas has traveled along the colon to reach the distal end of the bowel. The baby is put in an inverted position for a few minutes and a coin or a small barium paste is put at the possible site of the anus. The distance between the radio-opaque mark and the distal gas shadow is measured. If the

distance is more than 1 cm, this denotes high anomaly, while if less than 1 cm, a low anomaly is diagnosed.

- Urine is examined for the presence of meconium, if present; this denotes a fistulous communication to the bladder.

Surgical Treatment:

High anomalies are treated with.staged surgery. First stage is a temporary colostomy. Second stage is abdomino-ano-rectal pull-through. Third stage is closure of colostomy. Results are not usually satisfactory because continence may be weak. Low anomalies require only local perineal surgery. Results are excellent in the majority of patients.

Invertogram showing a high anorectal anomaly

Meconium plug syndrome:

This term is used to describe distal colonic obstruction associated with narrow left colon and thick plug of meconium in the distal colon. The baby is otherwise normal and gastrograf'in enema is usually curative.

Necrotizing enterocolitis (NEC):

This is the most serious and frequent gastrointestinal disorder of predominantly premature infants with a median onset of 10 days after birth. The incidence is increasing in view of the therapeutic advances in neonatal intensive care that allowed many more premature babies to survive.

It is characterized by necrosis, ulceration and sloughing of intestinal mucosa which frequently progresses to full-thickness necrosis and perforation. Gas-forming bacteria in the intestinal wall may lead to pneumatosis (air in the intestinal wall), a finding which is noted in plain abdominal radiographs in the supine or lateral positions.

Contrast studies are hazardous and may lead to perforation.

Treatment includes cessation of feedings, orogastric suction, systemic antibiotics and correction of hypoxia, hypovolaemia, acidosis and electrolyte disturbances.

The only absolute indication for operation is intestinal perforation.

At Laparotomy, the necrotic bowel is resected and the proximal bowel is made into a stoma as a primary anastomosis may not be safe. Late sequelae include short bowel syndrome due to extensive resections or intestinal strictures as a result of the healing process.

Childhood tumours:

Tumours in children show major differences from adult tumours. They are less common, show different cell types, and totally different biological behaviour. In general, tumours in children show better response to combination of surgery, chemotherapy and radiation. As a result, childhood tumours have better prognosis than adults tumours.

Common tumours in children (in order of frequency)

1. Acute lymphocytic leukaemia.
2. Brain tumours.
3. Neuroblastoma.
4. Nephroblastoma.
5. Hepatoblastoma.

6. Rhabdomyosarcoma.
7. Sacrococcygeal teratoma.

Common tumours in different age groups:

Neonatal period:

- Ovarian cystic lesions.
- Metastastasing neuroblastoma (curiously with excellent prognosis).
- Sacrococcygeal teratoma.

0-2 years:

- Nephroblastoma (Wilms' tumour).
- Neuroblastoma.
- Hepatoblastoma.

2-6 years

- Rhabdomyosarcoma of the bladder or prostate.
- Ovarian teratoma.
- Malignant lymphoma.

Clinical features of abdominal tumours in children:

- Abdominal mass. This is the commonest presentation especially in Wilms' tumour and neuroblastoma.
- Some tumours present by systemic manifestations as fever, anorexia and failure to thrive. A common example is neuroblastoma.
- Some neoplasms present by neurological symptoms as paresis or incontinence, e.g. neuroblastoma and rhabdomyosarcoma.
- Pain may occur due to metastases or torsion of an ovarian cyst.